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ALUMNI JOURNAL

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MORRIS L. BERMAN O.D.
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of The Massachusetts College of Optometry

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This edition initiates a new policy in the quarterly publication of the Alumni Association of M.C.O. It is our intention to include timely, informative articles of interest to the practicing optometrists, in addition to newsworthy items of alumni and school activities. The name has been changed to the *Alumni Journal* to reflect this change in policy. The editorial staff welcomes all comments and suggestions regarding the new format, contents, and contributions of news items concerning alumni as well as publishable articles. This is your journal which, with your help, can serve as a forum for discussions, opinions, evaluations of various clinical tests and procedures or any topics of mutual interest.

Message from Dr. Baldwin, President

The building fund received a major boost on May 20th when Mr. Edward Winston, President of the New England Optical Wholesalers Association, presented a check for \$8,500 to President Baldwin. This amount represents the first of three annual contributions from the individual firms comprising the membership of NEOWA. Their combined pledges total \$25,300. The presentation was made during the regular semi-annual meeting of the group, held for the first time in its history at the College.

After a business meeting, Dr. James Walters of the MCO faculty discussed new testing procedures in optometric examinations and demonstrated procedures now available in the Clinical Visual Physiology Laboratory.

Following the meeting, members of NEOWA and the MCO faculty and administration dined at Joseph's.

The contribution of the member firms of NEOWA to the MCO building fund represents a firm commitment to our future. We deeply appreciate their generosity and their continual interest in our programs. Cooperation between all groups who provide vision care is essential if the public is to be well served. We pledge our cooperation in this enterprise.

Following is a list of member firms of NEOWA:

Atlantic Optical Company	Merrimack Optical Company
Berks Optical Company	Metropolitan Optical Company
Budd-Pilgrim Optical Company	New England Optical Company
Chaffin Optical Company	Northeast Optical Co., Inc.
General Ophthalmic Corporation	Peerless Optical Company
Lawrence Optical, Inc.	Precision Optical Company
F. H. McGary Optical Company	Regal Optical Company
McLeod Optical Company, Inc.	Shawmut Optical Company
Tat-Fairfield Optical Co.	

We have just received word that a grant submitted jointly by MCO and Boston University Medical School to the Massachusetts Commission for the Blind and HEW has been approved for funding. This grant will support the low vision clinical activities of both organizations and permit co-operative programs in patient care, in the training of students and in continuing education. With our portion of the funds, the MCO Rehabilitative Clinic will be able to provide more complete staffing and will make available a wider array of visual testing equipment and visual aids. We invite optometrists to refer low vision patients for either complete work-ups or consultative services.

Commencement Exercises of the Massachusetts College of Optometry

The 1972 Graduation Exercises of the Massachusetts College of Optometry were held on Sunday, June 4th, in the New England Life Hall, Boston, Massachusetts.

Dr. Hyman R. Kamens, Dean of the College, greeted all in attendance and wished the graduating class well.

Irwin M. Nathanson, Class Valedictorian, expressed on behalf of his classmates the tremendous feeling of responsibility they all had towards the public as well as the profession.

The Honorable Kevin B. Harrington, President of the Massachusetts Senate, was the principal speaker. His pertinent remarks on vital issues were appreciated by those in attendance.

Dr. Maurice H. Saval, Trustee, then read a Citation for Senator Harrington. Dr. William R. Baldwin, President, and Dr. Richard W. Baker, Chairman of the Board of Trustees of the Massachusetts College of Optometry, presented Senator Harrington with the Honorary Degree of Doctor of Humane Letters.

Senate President Harrington, born in Salem, Massachusetts, is married to the former Kathleen Carney of St. Louis, Missouri, and has five children—two boys and three girls. He is a member of the Board of Trustees of the Newman Preparatory School; Member of the Board of Trustees of Salem Hospital; Member of the National Advisory Council of Hampshire College; Trustee of the Karin Grunebaum Cancer Research Foundation; Member of the National Committee for Support of the Public Schools; Member of the Massachusetts Advisory Council for the Deaf; and served as Chairman of the 1968-69-70 United Cerebral Palsy Campaign for the North Shore. He is now serving as President of the Senate; Chairman of the Committee on Rules; Chairman of the Committee to Reform Inter-

nal Workings of Legislature and a Member of the Special Committee on Electronic Data Processing.

Dr. John H. Carter, Professor at the Massachusetts College of Optometry, read a Citation for Dr. William W. Policoff. Dr. Baldwin and Dr. Baker presented Dr. Policoff with the Honorary Degree of Doctor of Ocular Science. Dr. Policoff has been a talented musician; a fine teacher at the undergraduate college level; and a scholar with earned degrees in law, veterinary medicine, pharmacy and optometry. As an optometrist, Dr. Policoff divided his efforts between caring for his patients, scientific study, development of new ophthalmic materials and furthering a spirit of good will between the professions of optometry and ophthalmology.

Dr. Hyman R. Kamens, Dean, read a Citation for Dr. Samuel J. Wasserman. Dr. Baldwin and Dr. Baker presented Dr. Wasserman with the Honorary Degree of Doctor of Ocular Science.

Dr. Wasserman was born in Boston and received his primary education in that city. He obtained his Bachelor of Science degree from New York University; a Master of Arts degree in education from Boston University; and a Doctor of Optometry degree from the Massachusetts College of Optometry. Dr. Wasserman has been associated with the Massachusetts College of Optometry for over twenty-five years as a member of the faculty. He has served on many committees and in many organizations in and out of optometry. At present, he is working with the rehabilitation of low vision patients and those patients in the General Clinic of the Massachusetts College of Optometry.

Forty-four graduates received the degree of Doctor of Optometry from President Dr. Baldwin and Board Chairman Dr. Baker.

The graduates are listed as follows:

CHARLES WILLIAM BALCH, JR., B.S., O.D. West Haven, Conn.
DANIEL EDWARD BAUSCH, B.S., O.D. Corona, N.Y.
LAWRENCE JOSEPH BERBRIER, O.D. Montreal, Canada
GERALD BLUMBERG, B.S., O.D. Valley Stream, N.Y.
JEROLD JAY BRODIE, B.S., O.D. Boston, Mass.
JACK BENJAMIN BRUMBERG, B.S., O.D. Miramar, Fla.
ANTHONY A. CAVALLERANO, B.S., O.D. Harrison, N. Y.
RONALD MARK CLINE, B.S., O.D. Randolph, Mass.
WILLIAM M. DELL, B.S., O.D. New York, N.Y.
MICHAEL STEVEN DIAMOND, B.S., O.D. New York, N.Y.
STEVEN GARY FELL, B.S., O.D. Brooklyn, N.Y.
STEPHEN ARTHUR FELTUS, B.S., O.D. West Acton, Mass.
DAVID MARVIN FINKELSTEIN, B.S., O.D. Qucens, N.Y.
DAVID L. FRIEDMAN, B.S., O.D. Baldwin, N.Y.
JOSEPH ALFRED GLEASON, B.S., O.D. Seneca Falls, N.Y.
RICHARD ALEXANDER GRAVES III, B.S., O.D. Presque Isle, Me.
DOUGLAS STUART HANCOCK, B.S., O.D. Casco, Me.
JOSEPH DAVID HASHIM, B.S., O.D. Pittsfield, Mass.
ANTHONY IANNUCCILLO, B.S., O.D. Larchmont, N.Y.
ARKADY KRAMAR, B.S., O.D. Springfield, Mass.
RALPH JOSEPH LEVOY, B.S., O.D. Braintree, Mass.
NORMAND ROGER MADORE, B.S., O.D. Lewiston, Me.
RICHARD CHARLES MARTINO, B.S., O.D. Marlboro, Mass.
CHARLES R. MITCHELL, JR., B.S., O.D. Sumter, S.C.
IRWIN M. NATHANSON, B.S., O.D. Jamaica, N.Y.
JEFFREY SETH NYMAN, B.S., O.D. Albany, N.Y.
NEAL N. NYMAN, B.S., O.D. Albany, N.Y.
BRIAN MICHAEL O'DONNELL, B.S., O.D. Marion, Mass.
PATRICK FRANCIS PHELAN, B.S., O.D. Inwood, N.Y.
ROBERT LEONARD PLATT, B.S., O.D. Waterbury, Conn.
PETER CARL PRUPAS, O.D. Montreal, Canada
RICHARD H. P. ROBILLARD, B.S., O.D. Lowell, Mass.
BOB MARTIN ROSEMARK, B.S., O.D. Milton, Mass.
BRIAN PAUL ROY, B.S., O.D. West Brookfield, Mass.
NORMAN JERALD SAVAGE, B.S., O.D. Sharon, Mass.
DAVID J. SCHURGIN, B.S., O.D. Brockton, Mass.
ARNOLD SHAPIRO, B.S., O.D. Baldwin, N.Y.
ROBERT ALLEN SMITH, B.S., O.D. Medford, Mass.
STANLEY DAVID STEINIK, B.S., O.D. Staten Island, N.Y.
HENRY SZIKMAN, O.D. Montreal, Canada
ROBERT G. TAMSETT, JR., B.S. O.D. Canajoharie, N.Y.
STEVEN JEROLD TISHLER, B.S., O.D. Hartford, Conn.
RICHARD J. WEINSTOCK, B.S., O.D. Forest Hill, N.Y.
JEFFREY A. WESSON, B.S., O.D. New York, N.Y.

In his Charge to the Graduating Class, President Baldwin reminded them of their responsibility in maintaining the highest levels of professional and academic excellence. Dr. Baldwin elaborated on the clear meanings of the educated and well-read individual. He warmly congratulated the Class of 1972 and on behalf of the College Faculty, Trustees and Administration wished them good luck and success.

A reception was held, following the graduation, in the Charter Room. The hostesses who prepared and served the refreshments were: Mrs. Honey Baldwin; Mrs. Beverly Bleck; Mrs. Betsy Christensen; Mrs. Barbara Kamens; Mrs. Margaret Lovell; Miss Valerie Molinaro; Mrs. Flo Schwartz and Mrs. June Wallis.



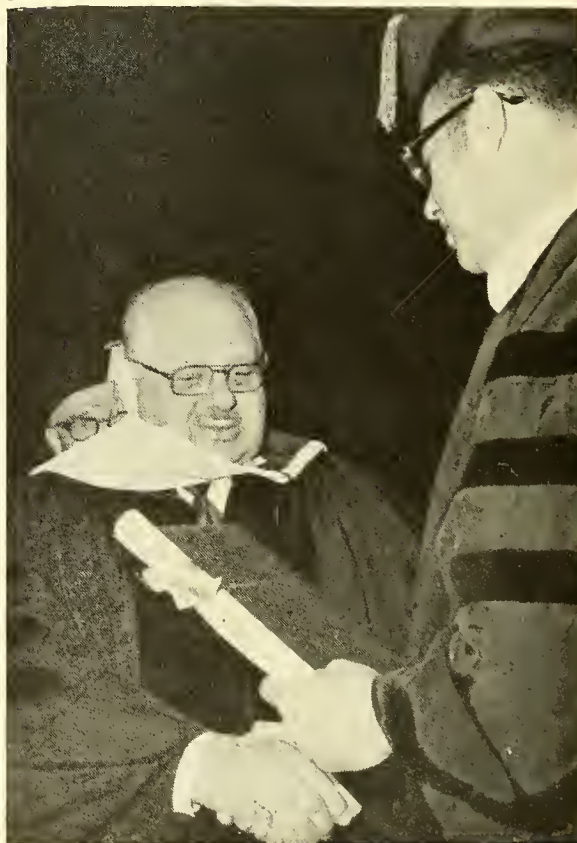
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1 Senate President Kevin B. Harrington addressing those attending Commencement Exercises.



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2 Dr. William R. Baldwin leading the Commencement Procession.

3 Dr. William W. Policoff receiving the Honorary degree of Doctor of Ocular Science from Dr. William R. Baldwin.



*From left to right:
Dr. Maurice H. Saval reads the citation for
Senator Harrington.
Senate President Kevin B. Harrington
Dr. William R. Baldwin*



*Dr. Samuel J. Wasserman receiving the Honorary
Degree of Doctor of Ocular Science from Dr.
William R. Baldwin.*

1972-1973
Academic and Clinical Calendar
for the
First, Second and Third Year Students

Fall Semester

5 September	Registration and tuition due for 2nd and 3rd year students.
6 September	Instruction begins for 2nd and 3rd year students.
7-8 September	First year registration and orientation. First semester tuition payment due.
11 September	Instruction begins for 1st year students.
18 September	Religious Holiday—No Classes.
2 October	Clinical program begins at General Clinic for 2nd and 3rd year students.
3 October	Follow Monday's schedule (Classes and General Clinic).
9 October	Columbus' Day — No Classes — General Clinic closed.
23 October	Veterans' Day — No Classes — General Clinic closed.
25 October	Follow Monday's schedule (Classes and General Clinic).
23-24 November	Thanksgiving Recess — General Clinic closed.
19-20 December	Registration for Spring Semester.
21 December	Classes end—General Clinic closes at 6 p.m.
22 December	Final Exams in terminating courses.

Spring Semester

8 January	Class and General Clinic resume — Tuition due on or before this date.
16 February	Vacation begins — Classes suspended and General Clinic closes at 6 p.m.
26 February	Classes and General Clinic resumes at 8 a.m.
8 April	Commencement.
13 April	Vacation begins — Classes suspended and General Clinic closes at 6 p.m.
25 April	Classes and General Clinic begins at 8 a.m.
8 May	Classes end and General Clinic ends at 6 p.m.
10 May	Final Exams begin.
18 May	Final Exams end.
4 June	Volunteer General Clinic for qualified first, second, third year students.
22 June	Volunteer General Clinical program ends at 6 p.m.

Fourth Year and Special Program Student Calendar

1972-1973

10 July	1st Session begins at 8 a.m.
4 September	Labor Day — Clinics closed.
29 September	1st Session ends at 6 p.m.
2 October	2nd Session begins at 8 a.m.
9 October	Columbus' Day — Clinics closed.
23 October	Veterans' Day—Clinics closed.
23-24 November	Thanksgiving Recess — Clinics closed.
22 December	2nd Session ends at 6 p.m.
8 January	3rd Session begins at 8 a.m. (remaining tuition due)
19 February	Washington's Birthday — Clinics closed.
30 March	3rd Session ends at 6 p.m.
2 April	4th Session begins at 8 a.m.
8 April	Commencement.
16 April	Thanksgiving Recess—Clinics closed.
23-24 April	National Board Exam — Clinics closed.
28 May	Memorial Day—Clinics closed.
22 June	4th Session ends at 6 p.m.

CALENDAR OF EVENTS

July 10 to July 14, 1972

Developmental Workshop with Dr. Jerome
Rosner

August 7 to August 18, 1972

Optometric Assistants' Program

The Annual Awards Banquet

The First Annual Awards Banquet, jointly sponsored by the Massachusetts College of Optometry and Alumni Association, was held on Saturday evening, June 3, at the Fensgate, in Boston.

Close to three hundred people attended, including College faculty, trustees, administrative staff, alumni, students and invited guests.

Dr. William R. Baldwin, President, greeted all present and turned the meeting over to Dr. Irvin Borish, who served as Toastmaster. His jovial comments were greatly appreciated by all.

Dr. Foster Namias, Chairman of the Committee on Awards, was then called upon to make the following presentations:

The Massachusetts College of Optometry Clinic Award was presented to Stephen Arthur Feltus.

This award is presented by the clinical faculty of the Massachusetts College of Optometry to that member of the graduating class who has achieved

superior competence in patient care by the sound clinical application of knowledge in the visual sciences and through example has demonstrated an exceptional sense of professional responsibility.

The Daniel Kuperstein Memorial Award was presented to Irwin M. Nathanson.

This award of \$100 is made available by the Kuperstein Family to the member of the graduating class who has achieved the highest scholastic average in Ophthalmic Optics.

The Valedictory Award was presented to Irwin M. Nathanson.

This award is made available annually by the faculty of the College to that member of the graduating class who achieves the highest general average in the courses of the professional curriculum.

The Beta Sigma Kappa Silver Medal Award was presented to Douglas Stuart Hancock.

From left to right:

Normand R. Madore, recipient of the Frederick E. Farnum Alumni Award; Dr. Glen Gulezian; Ralph J. Levoy, recipient of the Alumni Association Award, Frederick E. Farnum Alumni Award, and the Dr. Edward J. Troendle Jr. Award.



From left to right:

Dr. William R. Baldwin, Dr. Irwin M. Borish, Dr. G. Burt Holmes



The Beta Sigma Kappa International Honorary Society awards a silver medal annually to that member of the graduating class who has the most outstanding record of scholarship.

The Dr. Ralph H. Green Gold Medal Award was presented to Irwin M. Nathanson. This award is made available annually by Dr. Domenic V. Capone to the member of the graduating class who has achieved the highest scholastic average in Physiological Optics.

Dr. Borish then called on Dr. Glen Gulezian, President of the Alumni Association. Dr. Gulezian congratulated the new members of the Alumni Association and welcomed them into the profession. He pointedly stressed the urgent need, both in the Alumni Association and in the profession, for these young people to participate in and assume active roles in advancing the profession.

Dr. Gulezian then made the following presentations:

The Alumni Association Award was presented to Ralph Joseph Levoy.

The Alumni Association of the College offers an Alumni Plaque annually to that member of the graduating class who has achieved an outstanding scholastic and extra-curricular record.

The Frederick E. Farnum Award was presented to Ralph Joseph Levoy and Normand Roger Madore.

The Alumni Association of the College makes

available annually an award of \$100 to that member of the graduating class who demonstrates the highest degree of proficiency and expertise in contact lens practice.

Dr. Gulezian then introduced Dr. Paul Montminy, Vice-President of the Alumni Association. Dr. Montminy welcomed all of the new Alumni and presented each member of the graduating class with an Alumni Certificate.

Dr. Borish then introduced Dr. Arthur Jankolovits, representing the Class of 1970. Dr. Jankolovits noted the recent tragic and untimely death of his classmate, Dr. Edward Joseph Troendle, Jr.

Dr. Jankolovits announced that, in accordance with the wishes of his fellow-classmates, the Class of 1970 Gift Fund Award which would now be called the Dr. Edward Joseph Troendle, Jr. Award, was presented to Ralph Joseph Levoy.

Neal Nyman, Jeff Nyman, and Bill Dell, members of the graduating class, presented some interesting and rather unusual awards to various members of the faculty.

Dr. William R. Baldwin, President, on behalf of the faculty, made some very interesting observations on the graduating class. He congratulated the members of the class and welcomed them into their new profession.

All of those attending enjoyed a delicious meal and it was felt by all that the evening's festivities were a notable success.

M. C. O. ALUMNI IN NEW JERSEY

Meet at Luncheon during N. J. O. A. Congress

A Luncheon Reunion of M. C. O. alumni was held on Saturday, June 3rd, 1972 on the occasion of the New Jersey Congress. The reunion was suggested and implemented by Dr. Sol Lestch, '32, who contacted all alumni in the area. The response was good considering the fact that this is a first get-together of alumni in any area outside New England.

The twenty guests at the Luncheon were greeted and welcomed by Dr. Wilson, newly elected President of the N. J. Optometric Association.

Among those who attended were:

Dr. and Mrs. Sol Lestch; Dr. and Mrs. Feldman; Dr. and Mrs. Bloom; Dr. and Mrs. Goldstein; Dr. Bram; Dr. Milton Lebson; Dr. and Mrs. M. Ingram; Dr. and Mrs. Kurland and son; Dr. Katz and son.

It is planned to hold these reunions again. Any alumni who wish to participate are urged to write to Dr. Lestch or to Dr. M. L. Berman, Executive Secretary of the Alumni Association, M. C. O.

Summer Training Program For Optometric Assistants

This 10-day program was designed for the assistant who is now working for an optometrist but who could benefit from an intensive program of study to add to existing office skills.

The class, which was limited to twenty assistants, met for two weeks, Monday thru Friday, and used the teaching facilities of the main College building and the General Clinic.

Students were sponsored by their employing optometrists. The tuition fee for the 10 days of instruction for the assistant was \$125. Students brought their own office uniforms, if customarily used. Dormitory services were arranged.

COURSE OUTLINE

1. **OPHTHALMIC MATERIALS** — Basic Optics; Type of Ophthalmic Lenses (single vision, bifocals, trifocals, tints, plastic, occupational, etc.); Ordering (forms, laboratory relations); Verification (lensometry, lens clock); Facial Measurements; Frame Selection (optical, cosmetic and occupational factors). Total time in this phase—40 hours. A maximum of experience in learning at first-hand how to do with a minimum of lecture. Students had the opportunity to practice their skills in the working Clinic.

Instructor: David A. Greenberg, B.A., A.A., (Ophthalmic Dispensing, Erie Community

College), Licensed Ophthalmic Dispenser, N.Y. State.

2. **CLINICAL TECHNIQUES**—Visual Acuity Measurement (correct method of recording distance and near V.A.'s with and without prescription); Determination of Preferred Eye (sighting technique); Visual Skills Screening (complete Keystone battery of test cards, recording results); Visual Field Mapping (basic tangent screen and targets, Autoplot, recording perimeter); Care of Instruments and Examination Room Control.

Total time in this phase was 40 hours. Again, the teaching was heavily practical, and students had the opportunity to practice these techniques with patients during the normal working of the Clinic.

Instructor: Mary Scott, O.D., Instructor in Optometry, Coordinator of the Massachusetts College of Optometry and Fisher Junior College Optometric Technicians Program.

The total program was geared to 'doing' and learning the skills and techniques. At the end of the program, students were evaluated for skills learned. A certificate was issued upon satisfactory completion of this course.

On Sunday, May 7, 1972, a special educational seminar was held for members of the Loyalty Group of the Massachusetts College of Optometry. This seminar was a form of appre-

9:30 – 10:00 A.M. Coffee; Meet and Greet; M.C.O. Cafeteria.

10:00 – 11:15 Charles F. Mullen, O.D.; Director of Clinics and Assistant Clinical Professor of Optometry

"Pathology Review": brush-up and add-to your knowledge of primary ocular disease detection, and the signs and symptoms of systemic diseases exhibiting early ocular change.

11:15 – 12:30 P.M. Jerry A. Christensen, O.D., Ph.D., Assistant Professor of Physiological Optics and Optometry

"The Visual Fields": review the theory of visual field techniques and the significance and identification of lesions manifested in the visual fields.

ciation for their significant contribution to the M.C.O. Building Fund. Dr. Norman Wallis, Director, Division of Special Studies, arranged the following program:

1:30 – 2:45 Marshall V. Mark, O.D., D.O.S., Associate Professor of Optometry

"Pediatrics in General Practice": important considerations in caring for the young patient in the general practice of optometry, and how this patient population can grow in importance in your practice.

2:45 – 4:30 James W. Walters, Ph.D., Assistant Professor of Physiological Optics and Psychology

"The Clinical Use of Electrophysiology": the basic theory of the ERG and VER and the implications and usefulness of these techniques for optometry: a demonstration of these techniques in the Electrophysiology Clinic of M.C.O.

The Massachusetts College of Optometry was one of many colleges, universities and business organizations that were represented at the recent NEW ENGLAND OPPORTUNITIES DAY FOR MILITARY PERSONNEL AND VETERANS. This event was held recently at Fort Devens, Massachusetts and attended by men and women from veteran ranks in the area as well as from local military installations including Quonset, Pease, Otis, Newport and Westover.

Three students from the Massachusetts College of Optometry participated in this event, Ronald R. Ferrucci, '74, Joseph J. Rowey, '74, and James Pearson, '73. Ron Ferrucci is President of the second year class and, with Joseph Rowey and other members of the second year class, has done a commendable job in student recruiting. Jim Pearson is Student Council President and has participated in many extra-curricular activities for the benefit of the College.

The Dynamics of Visual Acuity

Eugene A. Craig, Ph.D

ABOUT THE AUTHOR

Dr. Eugene A. Craig has been Professor of Psychology at M.C.O. since 1969. Before coming to M.C.O., he worked as a Human Factors Engineer at the Navy Electronics Laboratory, performed research on training methods at the Human Resources Research Office in Washington, D.C., and taught at several educational institutions including Lehigh University and California State College of Pennsylvania. He has published in such areas as visual perception, training methods and creativity. He is currently writing a book titled, "Illusions, Hallucinations and Reality."

In an attempt to get students to realize that the seemingly simple task of measuring visual acuity involves complex processes, I have sometimes asked them to make a list of all of the variables they can think of that might influence such a measurement. These lists are impressive for their length and variety. They include such factors as the optical characteristics of the various transparent media of the eye, the intensity and spectral characteristics of illumination, physiological limitations such as the 'grain' of the retinal mosaic, and a variety of additional considerations ranging from glare to the ability of the patient to pay proper attention to the task.

Making such a list is likely to bring home to the student the need for careful control of testing conditions and the use of precise procedures if he is to obtain accurate acuity measurements. In addition, it is but a step from this to emphasize that no function with which the optometrist deals is truly 'simple'; that each involves complex interactions of many factors and each requires the same careful attention to measurement procedures if misleading information is to be avoided.

Often this discussion leads to the further question of how visual acuity is generated within the visual system; that is, what fundamental processes are involved when a patient reads a Snellen letter or responds correctly to some other acuity test object?

Identifying such processes and evaluating their interactive effects has interested me for several years. My interest has not, however, been primarily clinical in nature. As a psychologist rather than an optometrist, I have been concerned with understanding visual acuity as a perceptual threshold measurement which reflects the effectiveness of visual discrimination.

I first became concerned with these problems while I was working with perceptual effects that occur during prolonged visual fixation. Dr. Wolfgang Koehler, one of the founders of the Gestalt School of Psychology, had pointed out that if one maintains visual fixation for a minute or so without shifting one's gaze, several interesting things happen. One of these is that figures in the visual field during the fixation period appear to produce a kind of localized adaptation or figural after-effect that influences the apparent size and location of subsequently viewed visual figures.

The index of the extent of these figural after-effects used by most investigators at that time was to measure the apparent shift in location of test objects. It occurred to me that perhaps visual acuity might be a more sensitive index. I reasoned that visual acuity, as a threshold measure, should readily reflect any change in the dynamics of the visual system.

This idea was tested in an experiment. The subjects visually fixated the center of a black cir-

cle drawn in India ink on white cardboard. This fixation was maintained for three minutes. Then the black circle was quickly removed and a broken-line acuity test figure was substituted. Sure enough, when the acuity was measured for the area enclosed by the circle, it was found to be poorer than acuity for the same area measured without previous fixation of the circle. The prior visual fixation had produced some type of fatigue or satiation resulting in the decrement in visual acuity.

I continued working with various effects of prolonged visual fixation and noted, as had others, that during the period of fixation, objects within the visual field periodically disappeared and reappeared. The rate at which these cycles of invisibility and visibility occurred also seemed to differ depending upon their location within the visual field.

What was happening here? At the time—about twenty years ago—no one understood this phenomenon. I and a colleague, Malcolm Lichenstein, tried to find some answers. In one experiment, we set up a testing situation which involved having subjects visually fixate an "X" at one end of a black line.⁽¹⁾ No other figures were present in the visual field. The line could be adjusted so as to be in any orientation in the frontal-parallel plane relative to the subject that we desired to test. We then tested, in random order, all possible orientations of the line in 15 degree steps.

The subjects were instructed to maintain visual fixation and to press a telegraph key whenever the line, or any part of it, disappeared from view and to release the key as soon as it reappeared. The results were consistent and striking. They plotted into a sinusoidal curve showing maximum disappearance rates at the major axes of space (0° , 90° , 180° , 270°) and minimum rates at oblique angles (45° , 135° , 225° , 315°). We proposed as an explanation for the disappearances and for the variation with angle of orientation that an adaptation process was involved which, when complete, results in the complete disappearance of visually fixated objects. This adaptation process, though, was, we suggested, modified by involuntary eye movements. These eye movements differed in extent for different orientations and acted to relieve the adaptation process by changing the exact position of the line on the retina.

That our explanation was correct was shown by Riggs and his co-workers at Brown Univer-

sity⁽²⁾ and by Ditchburn in England.⁽³⁾ These investigators did not rely on our simple technique of instructing the subjects to attempt to maintain constant visual fixation. Instead, they were able to greatly reduce the effect of both voluntary and involuntary eye movements on the adaptation process. They achieved this by employing optical systems which served to maintain the retinal image in a stationary position in spite of eye movements. Riggs, for example, had his subjects wear a contact lens on which a tiny mirror was mounted. The image of a dark line on a bright background was reflected from this mirror onto a screen in front of the subject. A system of additional mirrors in the return of the image from the screen to the eye formed a compensating path such that whenever the subject moved his eyes the image followed precisely. In this way, the image remained stationary on the retina. Under these conditions the stabilized retinal images quickly disappeared from awareness. This verified the general hypothesis that adaptation processes underlie seeing which are of such a nature that, unless the visual image is allowed to move on the retina, it will soon disappear.

The question was then raised as to how these disappearances relate to visual acuity. When an entire figure drops out of view, it does not seem appropriate to blame this simply on a lapse of visual acuity. Instead, a more general characteristic of the visual system seems to be involved such that visual information that is unchanging, or redundant, eventually ceases to activate awareness. It seemed possible to many investigators, though, that visual acuity, in the sense of the resolving power of the visual system, still might be critically influenced by eye movements. It was thought that eye movements might be required in order for the retinal receptors to 'scan' a contour and that such scanning might be necessary for good visual acuity.⁽⁴⁾ Later investigations, particularly Keesey,⁽⁵⁾ have shown that this is not true. Visual acuity was found to be essentially identical for both a stabilized image condition and for a normal eye movement condition.

The fundamental processes upon which visual acuity depends are those involved in the formation of contours. That is, visual acuity can be considered to be the capacity to resolve, or discriminate, fine detail and these details are contours. If, for example, the break in a Landolt ring test object has been discriminated, the visual system has been successful in establishing sepa-

rate contours for the two sides of the gap. Perhaps the most fundamental of the interactions affecting visual acuity, then, are not the factors mentioned in the student's lists at the beginning of this article, but the interactions of contour processes with one another. This type of interaction largely accounts for the differences in legibility of different Snellen letters and for changes in visual acuity that occur when test letters or other test objects are isolated rather than being presented with other contours nearby.

The contour processes of any one figure appear to have a decremental effect on the establishment of additional contours within about four degrees of visual angle. This dynamic interactive process can be changed both in amount and direction by the design of acuity test figures and by the orientation of surrounding contours. This was demonstrated in an experiment in which I measured visual acuity under four conditions.⁽⁶⁾ Condition A involved a broken-line acuity test figure appearing alone in an otherwise homogeneous visual field. Condition B was modified by the addition of parallel lines on either side of the acuity test line. Condition C involved a change of relative orientation of these additional lines so that they

formed a 45 degree angle with the acuity test line. Condition D involved a further change of orientation such that the additional lines formed a 90 degree angle with the test line.

The results were clear-cut. Visual acuity was best for Condition A, next for B, then C, and finally D. The superiority of Condition A indicated—and this was supported by other studies—that visual acuity is most precise when no other contours are near. The other conditions show that this contour effect is directional in nature. It can be modified in amount by the relative orientation between the acuity line and other lines, with the greatest decrement appearing when contours of the additional lines are at right angles with the acuity line.

These studies have not settled all the problems of contour processes. It is important that such studies be continued. Additional investigations showing precisely how contours are established in the visual system should help us to understand, not just visual acuity alone, but give additional insight into a basic and important process that is required for the formation of our entire visual world.

REFERENCES

1. Craig E. A. and M. Lichtenstein, Visibility-invisibility cycles as a function of stimulus orientation. *Amer. J. Psychol.*, 1953, 64, No. 4, 554-563.
2. Riggs, L. A., F. Ratliff, J. C. Cornsweet and T. N. Cornsweet, The disappearance of steadily fixated visual test objects. *J. Opt. Soc. Amer.*, 1953, 43, 495-501.
3. Ditchburn, R. W. and B. L. Ginsborg, Involuntary eye movements during fixation. *J. Physiol.*, 1953, 119, 1-17.
4. Marshall, A. J. and S. A. Talbot, Recent evidence for neural mechanisms in vision leading to a general theory of sensory acuity. In H. Kluver (Ed.) *Visual mechanisms. Biological symposia. Vol. 7*, Lancaster, Pa.: Jacques Cattell, 1942.
5. Keesey, U. T., Effects of involuntary eye movements on visual acuity. *J. Opt. Soc. Amer.*, 1960, 50, 769-774.
6. Craig, E. A., Proximal figure effects on visual acuity. *Perceptual Motor Skills*, 1963, 16, 385-388.

We regret to hear of the death of another of our alumni, Dr. Hyman Laks,
315 West 70th St., New York City.

Dyslexia and the Role of the Vision Specialist

Paul J. Kantrowich, B.S., Class of 1974

DYSLEXIA

Hans Christian Anderson, Winston Churchill and Albert Einstein all shared a disability in common; it is said that these three famous individuals, from diversified fields, had exhibited many symptoms of "word blindness" either during their early schooling or during their lifetimes.¹ What is "word blindness" commonly referred to as dyslexia?

The lack of a consistent terminology has con- on the basis of its characteristics, etiology and to discuss the complexity of the problems it presents to both those who have it and those researchers seeking a solution to why it occurs.

The lack of a consistent terminology has continually contributed to a lot of unwarranted and unnecessary confusion in the field of dyslexia. The term "dyslexia" comes from Greek dys plus lexis. Dys means hard, bad or difficult; lexis pertains to words or to vocabulary of a language distinguished from its grammar.²

The term "dyslexia" alone is used variously in the literature of researchers to encompass both specific reading disability and also nonspecific secondary reading retardation. The term "specific developmental dyslexia" on the other hand is consistently used to designate or define a specific reading disability only. Among its synonyms in the literature I have researched are specific dyslexia, developmental dyslexia, primary reading disabilities, strephasymbolia, neurologic dyslexia, congenital word or letter blindness and Gestalt blindness.

Specific development dyslexia always has a neurologic basis—due either to genetic heritage or, on less frequent occasions, brain damage.³ As in the case of visual defects, neurologic deficits play a very significant role in cases of reading disability. An awareness of these neurologic factors is certainly of great importance to the ophthalmologists and optometrists and others concerned with dyslexia.⁴

On the other hand, nonspecific dyslexia—referred to by Rabinovitch as "secondary retardation"—may have a variety of causes; these include mental retardation, poor teaching, cultural disadvantage, environmental pathology, and a variety of other nonspecific factors—physical, emotional and educational—and the interaction between them. The concept that specific developmental dyslexia is by definition always a neurologic happening greatly simplifies differential diagnosis of reading disorders. The diagnostic objective becomes, in essence, to differentiate between specific dyslexia and nonspecific reading retardation. The differentiation has a direct practical bearing in that specific dyslexia calls for certain remedial reading techniques that are not essential in the management of nonspecific reading disorders. It is granted that the differential diagnosis between mild specific dyslexia and reading retardation often presents considerable difficulty. However, specific dyslexia in its moderate or severe forms should present no special problems in differential diagnosis. In a child of normal intelligence and without gross sensory impairment, the only condition that specific dyslexia in its severe form may be confused with is the dyslexia of conversion hysteria. This "pure" form of psychogenic dyslexia is extremely rare and is not likely to present much of a diagnostic dilemma.⁵

Dyslexia, as used to describe the disorder of the child, is not that offered by Eisenberg (1962); a reading disability not caused by sensory handicaps, mental deficiency, poor or deficient schooling and/or poor motivation. Other definitions are equally inappropriate for a youngster who is mentally retarded and has poor schooling experiences. Robbins offers as a definition of dyslexia

¹ Flower, R. M. *Reading Disorders*, page r.

² Thompson, L. *Reading Disability*, p. x.

³ Hellmuth, J., *Learning Disorders*, Vol. II, p. 27.

⁴ Hellmuth, J., *Learning Disorders*, Vol. III, p. 466.

⁵ Hellmuth, J., *Learning Disorders*, Vol. II, p. 28.

the impairment of the ability to read silently or in the absence of aphasia and independent of any speech defect. This definition of "aphasia" and "dyslexia" are mutually exclusive. Robbins also defines alexia as a type or form of sensory aphasia characterized by the impairment or loss of the ability to read silently although vision and intellect are unimpaired. This definition of dyslexia by Robbins includes alexia as a subclass of a specific form of aphasia.

OBJECTIVE CHARACTERISTICS OF DYSLEXIA

Various attempts have been made to identify and classify the errors which a dyslexic child makes when reading, spelling and writing. Among the valuable terms that have been placed into context to describe the various categories of errors and visuo-spatial difficulties manifested by static and kinetic reversals, correlating difficulties, directional confusion, difficulties in reauditorizing and revisualizing and poor word attack skills. The most frequent features of specific developmental dyslexia generally viewed as diagnostic include:

1. Failure to learn to read normally with conventional instruction, particularly with the whole-word technique. The disability may continue to persist into adult life and is often refractory even to remedial reading methods.
2. A variety of bizarre but consistent perceptual errors in reading, spelling and writing which in their severest form result in a total reading disability, or alexia, widely described as letter blindness and word blindness.
3. Strong familial incidence, apparently transmitted as a dominant genetic trait. However, dyslexia is also associated, though less frequently, with chronic brain syndromes.
4. Frequent association with a variety of minimal neurologic symptoms, pointing to a parieto-occipital dysfunction and neurophysiologic immaturity. Among these associated signs are crossed and confused dominance, failure to establish lateral dominance or ambidexterity.
5. Frequent association of specific dyslexia, particularly in the younger child, with the hyperkinetic behavior syndrome, with auditory imperception and with visuo-

perceptual and visuomotor impairments, including poor figure-background discrimination and spatial orientation.

6. Characteristic psychological test results (that will be discussed later in this paper).
7. Frequent occurrence of abnormal electroencephalograms, characterized by slow waves, or immature tracings and generalized dysrhythmias.⁶

Many specific deficits are illustrated by the following errors:

1. Confusion of reversible letters such as b-d-p-q, M-W, and n-u-v, the so-called static reversals, and confusion of letters with subtle graphic differences, such as h and n, v and y, l and I.
2. Confusion of letters with acoustic similarities, such as d and t, and v and f.
3. Inversions or transpositions of letters within a word in reading, such as "its" for "sit" and "three" for "there", including frank reading and writing, such as "was" for "saw" and "ton" for "not"; and in writing transposition such as "sopek" for "spoke", "frie" for "fire", and "lillet" for "little". These are the so-called kinetic reversals.
4. Additions and omissions of letters or sounds, such as "bother" for "brother", and "apt" for "about" in reading; and "noverd" for "nod", and "hugery" for "hunger" in writing.
5. Substitution of words with similar or related meaning, such as "cat" for "kitten", "quack" or "chicken" for "duck", "airplane" for "train", "road", "bike" or "horse" for "ride", "little" for "kitten", and "like" for "love".
6. Guessing at words from the first or last letters or syllables, such as "diesel" or "dentist" for "dress", as well as from context, such as "finest" for "richest", and "said" for "asked".
7. Inability to synthesize letters into syllables, and syllables into words, such as being able to read the syllables "el" and "bow" but not the word "elbow", or "col" and "or" but not the word "color".⁷

Many of the characteristics mentioned above also occur in the normal course of learning to read but Money points out "what distinguishes

⁶ Hellmuth, J., *Learning Disorders*, Vol. 111, p. 29.

⁷ *Ibid.*, page 30.

the child with specific dyslexia is the frequency and persistence of such errors well beyond the time at which they have become uncommon for the normal child".⁸

SOME NEUROLOGICAL ASPECTS OF DYSLEXIA: LESIONS

In evolving a theoretical construct, it is useful to review the semiautonomous systems concept of brain function. In 1963 Hebb provided a model that provided an excellent frame of reference to which we can consider the problem of aphasia in childhood. It purports that the brain comprises systems which are semiautonomous from one another. From this point of view, we might argue that there are many essentially independent systems.⁹ We must assume that dysfunction in the brain can occur and it may occur without seriously debilitating the patient. As in developmental dyslexia (Orton 1937), it is generally acknowledged that a child can sustain a disorder in auditory language without having first acquired this verbal system. Hermann (1959) has shown us evidence that dyslexia occurs on a familial or hereditary basis.

It is shown how 33 information-carrying channels can be conceptualized as combination channels through which information transfer flow takes place during learning and problem-solving. Each learning channel in turn, contains at least seventeen systems which operate upon information in that channel to transduce an input challenge into an output solution. The containing potential learning and problem-solving disabilities or disorders—total defects as well as partial defects or dysfunctions. Learning disabilities are viewed as an unexpected discontinuity or lesion in this emergence order. Screening techniques have been developed to trouble-shoot a total of 17 x 33 or 561 defect-type lesions in which systems are completely absent. Henry J. Mark contends there are nine types of lesions that are unambiguously identifiable by psychophysical curves now expanding potential lesions in the system to 5049.¹⁰

Khoudadoust, Schenker, Friedenwalk and Krebs (all independently) found incidences of retinal hemorrhaging in excess of 20% of children they examined who suffered from a brain dysfunction.¹¹ Walsh and Lindenberg have demonstrated the role of hypoxia relative to damage to the optic pathways. While relating reading retardation and minimal brain dysfunctions, it is

of importance to be aware of the six areas of anatomical importance in reading. Reading is first a matter of vision or sensation, and, secondly, interpretation or perception at what is seen. Images are recorded on the retina and transduced electrically to the optic nerve; then, via the LGB, to the calcarine fissure in the occipital lobe. This is the primary visual receptive area called the striate cortex (Area 17, Brodmann's Area). Surrounding area 17 are cortical areas 18 and 19 which are secondary and tertiary areas of vision. A lesion in this area 19 may impair the ability to read. The angular gyrus, located in the posterior part of the parietal lobe and lying adjacent to area 19 serves the same function. The angular gyrus is essential in both reading and writing. The area of Wernicke concerned with the recognition and recall of speech by its connections to the angular gyrus reinforces by auditory stimuli the ability to understand written language. Lesions of either Wernick's area of Broca's area complicate the learning of reading.¹²

Acquired dyslexia describes patients who have had the adequate ability to read but have lost it possibly due to head trauma, vascular accident of malignancy. In acquired dyslexia, many times there is a right hemiparesis and sometimes a right hemianopia. These are rare in children, however. The adaptability and flexibility of the brain within childhood developmental years facilitates compensations that are absent in the less flexible and rigidly set adult brain. Kawi and Pasamanick did a review of 100 children and concerned themselves with gestational history. In this study Pasamanick related most problems in children to what he terms "the productive casualty", i.e., the concept of some cortical or cerebral problem to the fetus or some nutritional or other supporting lack of prenatal care of the mother. He believed there was a biologic factor to dyslexia. In one of his cases, Pasamanick encountered severe dyslexia due to neurologic defects. The EEG of one of these dyslexics showed a focal lesion in the left parieto-occipital region. The size of the lesion and additional vascular difficulties suggested either a vascular shunt, hemangioma or meningioma. It turns out this patient had a congenital anomaly of arteriocovenous communication—he

⁸ *Ibid.*,

⁹ Arena, John, (Ed.), *Management of the Child*, p. 257.

¹⁰ *Ibid.*, p. 258.

¹¹ Hellmuth, J. *Learning Disorders*, Vol. III, p. 460.

¹² *Ibid.*, page 441.

has recovered totally and is completely illiterate.¹³

Another dyslexia had gross prenatal hypoxia at birth. He had periodic anoxic episodes with convulsions but recovered although he remained totally aphasic and absolutely alexic. His verbal IQ was 52 and performance IQ was 104 but he cannot talk sensibly. In this patient, anoxia led to a specific symptom pattern.

Roger Sperry feels that perhaps a "possible factor in dyslexia is an overly strong or extensive, perhaps bilateral, development of the verbal, major-hemisphere type of organization that tends to interfere with an adequate development of spatial gnosis in the minor hemisphere. The facts that general verbal capacity tends to be good in dyslexics and that the frequency of dyslexia is higher amongst left-handed persons would fit such an interpretation".¹⁴

VISUAL PROBLEMS: ROLE OF THE VISION SPECIALIST

Reading disability or disorder, as it concerns the ophthalmologist or optometrist, is a variable condition but this does not make it less real. The complexity of this entity has led to the application of many anomalies.

Positive ocular or ophthalmic findings are rare amongst those who cannot read, although ocular disorders may be found among slow, but accurate, readers.¹⁵

Some investigators such as Selzer and Blake and Dearborn appear to feel that vision is less adequate among poor readers or that more visual defects occur among them. On the other hand, Farris, Monroe and Stullken are of the opinion that deficient visual acuity is not particularly important as a cause of reading failure.¹⁶ Poor visual acuity is rarely a deterrent to learning to read even if the visual acuity is less than 20/200 due to a whole condition of variables that enter into the ability to see and the desire to learn. Refractive errors are probably of negligible importance also in causing reading disorders unless they go uncorrected. Refractive error and extrinsic eye muscles have no greater incidence among children with reading disabilities than among those without.³² Muscle imbalance and problems in convergence are possible causes of reading difficulties. Oculomotor problems do not interfere with the recognition of symbols. It is very difficult for a vision specialist to convince the parents of a cross-eyed child that the eyes are not the cause of reading disability; there will be sometimes a

token of agreement but in their hearts they know we are wrong.¹⁷ It is also true that abnormal occipital discharges on the EEG are often associated with amblyopia ex anopsia. This may be true, but it is noted that EEG records of children ages 5 to 6 are difficult to interpret anyway. Amblyopia is really part of a brain dysfunction, a central suppression, and therefore could be a manifestation of brain dysfunction. When caught early enough, suppression and the onset of amblyopia can be discouraged and possibly discarded. The treatment is varied and, for youngsters, centers around a patching technique and orthoptics.¹⁸

MANAGEMENT OF THE DYSLEXIC

Management, as in the diagnosis, is a threefold approach. It is directed toward the hyperkinetic behavior syndrome, the learning disorder and the emotional overlay.

For the hyperkinetic behavior, both a trial of behavior-modifying drug therapy and home management are strongly recommended. Dextro-drine, Ritalin, Mellaril, Benadryl, Deaner, Dilantin can be used in this psychotropic drug therapy. Large doses are avoided, particularly in the school clinic setting and the drug that is effective in the smallest dose is preferred. Tranquilizers such as Thorazine, Librium and Valium are used mostly for older children with very low frustration tolerance and hostile acting-out behavior. A consistent structured home environment stressing regularity in health habits is essential. This includes regular bedtime, mealtime and indoor and outdoor playtime, limited television viewing and responsibility for minor home chores. It is felt a structured routine helps the child become more structured and better organized. . . . thus all contributing to a better school adjustment.¹⁹

Learning disorders are nonspecific and are secondary to the hyperkinetic syndrome itself. Successful drug therapy usually results in immediate improvement in academic achievement and motivation. If specific learning disorders are present, such as specific dyslexia, then remedial reading therapy is absolutely essential. Depending on the severity of the specific disorder level, pro-

¹³ Keeney and Keeney, *Dyslexia*, p. 113.

¹⁴ Sperry, Roger, *Cerebral Dominance in Perception*, page 167 (SA printout).

¹⁵ Thompson, Lloyd, *Reading Disability*, p. 41.

¹⁶ *Ibid.*, page 44.

¹⁷ *Ibid.*, page 43.

¹⁸ Keeney and Keeney, *Dyslexia*, page 116.

¹⁹ Hellmuth, J. *Learning Disorders*, Vol. 11, p. 39.

grams range from normal school situations to special individually oriented programs.²⁰

In mild cases of emotional overlay, psychiatric referral is not usually indicated. They tend to subside without the benefit of psychiatric or psychological help; this is providing the hyperkinetic behavior and learning disorders are also being successfully dealt with. In older children, the emotional overlay resulting from chronic school failure is frequently very severe and psychiatric referral may be definitely indicated as an essential part of the total management program.²¹ General management would also include parental counselling, discussing in full the exact nature of the difficulties on a periodic basis.

SUMMARY

Reading is a psycho-physical process involving visual stimulation followed by transmission of neutral impulses to the brain. Cerebral acts follow which result in conscious perception of the words as objects and as symbols.²² When dealing with dyslexic children, one must remember that they come to the physician probably for just one reason, they cannot read. They come not because

they cannot romp and play like normal children, not because they have an unusual eye-movement or not because they are anything but non-readers. If, in the course of study, it is found they have other problems, they can be given attention for those disorders but the basic need for help is in learning to read regardless of the etiology. At present, the best therapy for the non-reader appears to be hard daily work in reading. This is slow and costly of time, effort and patience for both child and teacher. There is no substitute, however, for giving a child with a symbolization problem help in the specific area of his needs. Vision care specialists must insure the dyslexic has the optimum visual capacity possible, and that, if a child is suspected of having a dyslexic problem, he is tasked with the responsibility of properly referring the parents to an individual, as part of the interdisciplinary team concept, who can give this child a possible new lease on his future.

²⁰ *Ibid.*, page 32.

²¹ *Ibid.*, page 33.

²² Eames, Thomas, *Visual Handicaps to Reading*, Vol. 141 p. 17.

Bibliography

- Arena, John T., *Management of the Child With Learning Disabilities: An Interdisciplinary Challenge*, Association For Child With Learning Disabilities, Pittsburg, 1967.
- Benyon, Shiela, *Intermediate Programming For Slow Learners*, Charles E. Merrill Publishing Company, Columbus, Ohio, 1965.
- Eames, Thomas H., *Journal of Education, Visual Handicaps To Reading*, BU School of Education, Number 3, Vol. 141, Feb. 1959.
- Flower, R. M. (Ed.), *Reading Disorders*, F. A. Davis Company, Philadelphia, 1965.
- Hellmuth, Jerome (Ed.), *Learning Disorders*, Volumes II and III, Special Child Publications, Seattle, 1966.
- Johnson, Doris and Helmer Myklebust, *Learning Disability Education Principles and Practices*, Grune and Stratton, New York, 1968.
- Keeney, A. and V. Keeney, *Dyslexia*, C. V. Mosby and Company, St. Louis, 1968.
- Lanick, Nancy (Ed.), *Reading in Action*, Vol. 2, International Reading Association Conference Proceedings, Scholastic Magazines, New York, 1957.
- Schubert, Delwyn G., *Reading In Reading Practice Theory Research*, Thomas Y. Crowell Co., New York, 1968.
- Travis, L., *Handbook of Speech Pathology and Audiology*, C. V. Mosby and Co., New York, 1966.
- Thompson, Lloyd J., *Reading Disability: Developmental Dyslexia*, Charles C. Thomas Co., Springfield, 1966.

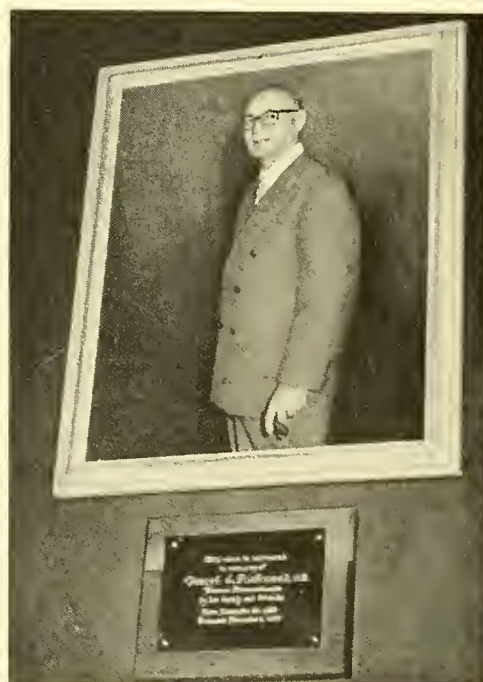
The Class of 1970 was saddened by the tragic loss of Dr. Edward Joseph Troendle, Jr. Ed passed away Wednesday, May 31, 1972 and we all will miss him.

On Saturday evening June 3, 1972, Drs. Philip and Arnold Richmond dedicated a portrait of their late father, Dr. Joseph C. Richmond, at ceremonies held at the Library of the Massachusetts College of Optometry. Present were members of the Board of Trustees, members of the Executive Committee of the Alumni Association and members of the Faculty. Members of the

Richmond family were present to see the unveiling of a portrait of Dr. Joseph C. Richmond and to hear Dr. William R. Baldwin speak of the contributions to the profession of optometry made by this man. Dr. Philip Richmond briefly spoke about his father and expressed his satisfaction that this portrait will be hung in the Library where his father spent a great amount of time.



The Richmond family at the unveiling of the Dr. Joseph C. Richmond portrait.



The Dr. Joseph C. Richmond portrait unveiled in the College library.

Drug Abuse

by David A. Vito, A.B., Class of 1973

Drug abuse is a growing problem today; one that cannot be ignored. As a future member of the health care field, it is of vital concern to acquaint oneself with the different types of drugs, their use, their long-range effects, and their potential for abuse.

According to the Bureau of Narcotics and Dangerous Drugs, drug abusers fall into three main groups:¹

- 1) Situational—a student may use pep pills to cram for exams, or the truck driver during long hauls.
- 2) Spree—used for 'kicks' or the experience.
- 3) Hard-core addicts—one whose activities revolve almost entirely around drug experiences and securing drugs.

Every individual is a unique combination of physical and emotional forces. Drug effects are complex, not completely understood, and frequently unpredictable. Drugs have somewhat different effects on different individuals; moreover on the same individual, a drug's effect can vary from one day to the next. No drug is harmless.

Abuse of drugs usually is a matter of personal choice—at first. But drug abuse can lead to drug dependance, "the repeated administration of a drug on a periodic or continuous basis", according to the World Health Organization's definition. Patterns of drug dependance differ depending

upon the nature and amount of the drug, the characteristics of the person taking it, the circumstances in which it is taken, and, not least, the expectations of the user.

There are some helpful hints to keep in mind when observing a patient, an acquaintance, or someday one's own children. It is reasonable to be suspicious if a young person suddenly starts to wear dark glasses all the time—even at night. This may be a way to hide the redness of the eyes caused by marijuana use or the dilated pupils due to the use of LSD. Narcotics users, by contrast, may have constricted, or pinpoint pupils. Those who take drugs by injection will have needle marks on their arms, legs, or other parts of their body. They may wear long sleeves to hide the 'tracks'. Sometimes injections leave oozing blood traces on the sleeve. Needless to say, none of these signs is proof of drug taking—but they may be signals!

The following table gives a general summation of a variety of drugs which are commonly used and abused. There are four major groupings:

- 1) sedatives
- 2) stimulants
- 3) hallucinogens
- 4) hard narcotics.

TABLE I

Name	Chemical Name	Slang Name	Medical Use
SEDATIVES Barbiturates	Phenobarbital (Luminal) Secobarbital (Seconal) Pentobarbital (Nembutal) Thiopental (Pentothal) Amytal Tuinals	Phennies Red Devils Yellow Jackets — Blue Angels Rainbows Goof-balls Barbs, Candy Downers Sleepers	Sedation, Relieve high blood pressure Epilepsy, Hyperthyroidism
STIMULANTS Amphetamines	Benzadrine Dexadrine Methadrine Desoxyn Methamphetamine Methedrine Phenmetrazine	Bennies Dexies Meth — Speed Ups Pep pills	Relieve mild depression, Control appetite, Promote wakefulness
HALLUCINOGENS LSD	d-lysergic acid diethylamide	Acid, Sugar Big D, Cubes Trips	Experimental study of mental funct. Alcoholism.
DMT	Dimethyl-triptamine	Businessman's High, AMT.	None.
Mescaline	3, 4, 5-trimethoxyphen- ethylamine	Mesc.	None.
Psilocybin	3 (2-dimethylamino)- ethylindol-4-ol-di-hydrogen phosphate.		None.
Marijuana	Cannabis sativa	Pot, Grass Reefers Mary Jane Hashish Tea, Gage	None in U.S.

HARD NARCOTICS

TABLE II

Name	Chemical Name	Slang Name	Medical Use
Heroin	Diacetyl-morphine	Junk, Horse Scag, Stuff Scat, Smack Harry, H.	Pain relief (not legally available in U.S.)
Morphine	Morphine sulphate	White stuff M.	Pain relief.
Codeine	Methyl-morphine	Schoolboy	Ease pain and coughing.
Methadone	Dolophine Amidone	Dolly	Pain relief Management of heroin dependance
Cocaine	Methylester of benzoylecgonine	Corrine Coke, Bernice Snow, Flake Star Dust Gold Dust	Local (surface) anesthesia

TABLE 1—(Contd.)

Mental Dependence	Organic Damage	Warning Symptoms	Dangers
Yes	Yes	Apparent drunkenness (sluggish confusion) Depression, Lethargy Quarrelsome, Aggressive Loss of Coordination Mental & Emotional Instability Overdose: coma.	Combined with alcohol can cause death. Intentional or accidental suicide by overdose. Severe withdrawal: dizziness, vomiting, fainting, tremors, delirium, coma, and possibly death.
Yes	Yes?	Excitation, Nervousness Talkativeness, Dry mouth Heavy perspiration Loss of appetite Sleeplessness Large doses: delusion, hallucination hostility, psychosis aggressiveness	Fatigue: substitute drug for needed rest. Impairs judgement May become combative Reckless behavior Coma, and possibly death.
No?	No?	**Vary greatly** Restlessness Inability to sleep Dilated pupils Hallucinations Distortion of sensory perceptions.	Possible permanent loss of sanity or personality changes. Flashbacks of the reaction occur mos. later. Generates impulses of violence and self-destruction.
No?	No?	Unpredictable behavior Decreased ability to discriminate between fact and fantasy.	Impaired judgment Possible chromosomal damage.
No?	No?	Panic or terror Psychotic reactions	..
No?	No?
Yes?	No	Effects vary with method of injection: Euphoria Altered concept of time and space Visual distortion and hallucinations Impaired judgment Exaggerated sense of well-being.	Can lead to aggressive behavior Altered perceptions can lead to accidents Can lead to serious drug abuse thru cont with pushers (to date facts show marijuana alone does not lead to physical dependence be possibly psychologic dependence).

TABLE 1—(Contd.)

Dose & How Taken	Duration of Effect and Effects sought	Long-term Symptoms	Physical Dependence
50-100 mg., Swallowed or Injected	4 hrs., Euphoria Reduction of anxiety.	Addiction with severe withdrawal symptoms Possible convulsions Toxic psychosis	Yes
2.5-5 mg., Swallowed or Injected	4 hrs., Alertness Activeness	Loss of appetite Delusions Hallucinations Toxic psychosis	No
100-500 μ g., Swallowed	10 hrs., Insight Distortion of Senses Exhilaration	May intensify existing psychosis Panic reactions	No
1-3 mg., Injected	1 hr., Insight, Distortion Exhilaration	???	No
350 μ g., Swallowed	12 hrs., Insight, Distortion, Exhilaration	???	No
25 mg., Swallowed	6-8 hrs., Insight, Distortion, Exhilaration	???	No
1-2 cigarettes Smoked, Swallowed, or Sniffed	4 hrs., Relaxation Increased euphoria Sociability Perceptions	???	No

TABLE II—(Contd.)

Dose & How Taken	Duration of Effect & Effect Sought	Long-term Symptoms	Physical Dependance
Varies, Injected or Sniffed	4 hrs., Euphoria, Prevent withdrawal discomfort	Addiction, Loss of appetite, Constipation	Yes
15 mg., Swallowed or Injected.	6 hrs., Euphoria, Prevent withdrawal discomfort	Addiction, Loss of appetite, Constipation	Yes
30 mg., Swallowed.	4 hrs., Euphoria, Prevent withdrawal discomfort	Addiction, Loss of appetite, Constipation	Yes
10 mg., Swallowed or Injected.	4-6 hrs., Prevent withdrawal discomfort	Addiction, Loss of appetite, Constipation	Yes
Varies, Sniffed, Injected or Swallowed.	Varies, short Excitation, talkativeness	Depression Convulsions Delusions Toxic psychosis	No

TABLE II—(Contd.)

Mental Dependence	Organic Damage	Warning Symptoms	Dangers
Yes	No	Constricted pupils Bruises on arms and legs (abuser usually wears long sleeves) Alienation, Detachment, preoccupation (with securing drugs)	Physical and psychological addiction Blood infections, tetanus abscesses, hepatitis and venereal disease Malnutrition
Yes	No	Lack interest in personal hygiene	Possible involvement in crime
Yes	No	Runny nose Loss of weight Itchiness	Withdrawal reactions are serious: vomiting, flu-like illness, running nose and tearing eyes, heavy perspiration, muscle spasm, aches and pains, altered blood pressure, pulse, respiration, and temperature, diarrhea.
Yes	No	„	„
Yes	Yes ?	„	Can ultimately cause death.

SEDATIVES

Sedative drugs are manufactured for medical purposes to reduce tension and anxiety, to treat certain psychosomatic disorders, and to induce sleep. Certain sedatives are used in the treatment of epilepsy.³ Barbiturates—one type of sedative—vary in duration, the short acting ones more likely to lead to abuse:⁷

Pentothal (fastest acting) . . . Nembutal . . . Seconal . . . to Luminal (slowest acting).

The principal response elicited by barbiturates is depression of the central nervous system, through action on the cerebral centers. In large doses they can depress the brain centers responsible for respiration's rhythmicity. Those who take excessive amounts of barbiturates usually go into a coma. In persons who are used to taking large doses, i.e., their tolerance level is high, barbiturates may produce restlessness, delirium, and excitement resembling the alcoholic.

Barbiturates are frequently used in conjunction with amphetamines (stimulants), often to induce sleep after the 'jag' is over. Alternatively, the amphetamines may be taken to counteract the barbiturate 'hangover'. A regular, heavy user who has built up tolerances to sedatives and requires larger doses to obtain the desired effects, will suffer withdrawal symptoms when the drugs are suddenly stopped. The severe withdrawal state resembles delirium. The user is agitated, restless, may have muscle cramps, nausea, and convulsions.⁵ In addition, he may see things that are not there, have dilusional, confused thoughts.

Death can occur when a number of barbiturates are consumed by someone who is intoxicated with alcohol. These drugs (alcohol is another form of sedative) act as synergists and are additive in their effects.³ This is also true of combinations of barbiturates with anesthetics, narcotics, or tranquilizers. These drugs act to intensify, or potentiate each other's effect.⁷

STIMULANTS

Stimulants are drugs, usually amphetamines, which stimulate the central nervous system. These drugs resemble the natural body hormones, epinephrine and norepinephrine, i.e., they mimic the hormones at the nerve endings. Amphetamines stimulate areas which control blood pressure, heart, respiratory, and metabolic rates, all of which are increased.⁸ Appetite is markedly decreased and the senses hyperalert, thereby putting the body in a general state of stress as if

threatened.⁶

Stimulants induce a transient state of well-being, self-confidence, and alertness. They are used to combat fatigue, curb appetite, reduce mild depression, and treat Parkinson syndrome.⁵ Stimulants include amphetamines (Benzedrine), dextroamphetamines (Dexedrine), methamphetamine (speed) and cocaine—though this is usually categorized with hard narcotics.⁸ Mild stimulants include coffee, tea, and caffeine.

Overuse of amphetamines may start in the physicians office, where they are prescribed for depression, obesity, or lethargy, or through various illicit channels. Abusers of amphetamine can be situational, for example, to stay awake while driving or studying, or to excel in an athletic contest.³ This type of abuse rarely leads to difficulties, though it may lead to a spree or binge. This abuser tries to counteract depression, to feel high, or to keep going. These individuals are likely to become drug dependant. Recently, another type of amphetamine abuser has developed, the speedfreaks or methheads. This person injects massive doses intravenously to produce practically the same effect as cocaine.⁶

In ordinary amounts, amphetamines provide transient sense of alertness, diminished appetite, heart rate increased, rapid breathing, dilated pupils, dry mouth, sweating and headache.⁸ These drugs however create a dependence, and as the tolerance increases, higher doses are required to obtain the original effect. A person on amphetamines has a tendency to talk rapidly and loud, pace around, perspire, and perform other stereotyped acts.⁵ He appears oversensitive to stimuli, anxious and jumpy, and may develop a mood of apprehension or panic. Speed can occasionally kill, from accidents during paranoid delusions, homicidal rages, or injection with contaminated substances.

HALLUCINOGENS

LSD is a manmade chemical, legally classified as hallucinogenic, i.e., mind affecting drug. The average dose of 'acid' is a tiny speck of colorless, odorless, tasteless material, with a lasting effect of 8-10 hours.³ The physical effects consist of enlarged pupils, a flushed face, chilliness, perhaps a rise in temperature and heart beat, with a slight increase in blood pressure.⁹

The psychological effects of LSD vary considerably according to the amount taken, the personality of the user and the circumstances

under which the drug is taken. The effects can be notably different at different times—even on the same person. Marked changes in sensation are typical. Vision is significantly altered; users are likely to see unusual patterns, and the meaning what is seen is often transformed. One sensory experience may be translated or merged with another; for example, smells may be felt, sounds may be seen. Illusions and hallucinations can occur, and delusional thoughts are sometimes expressed.⁹ Emotional variations are marked, ranging from bliss to horror, sometimes within the same experience.⁶

Users often refer to 'good' and 'bad trips', or 'bummers.' Because of the impaired time sense, such an experience can assume the proportions of a terrible nightmare from which one cannot return. He doesn't know how much time is passing, but he does remain conscious. After the drug wears off, the user usually remembers much of what has happened to him.

Just how LSD works in the body is not yet known. It seems to affect the levels of certain chemicals in the brain, like norepinephrine, and produce changes in the brain's electrical activity.⁹ There is no indication that LSD is physically addictive, but its effect on the mind is entirely unpredictable.⁵ A definite danger reported in recent clinical reports is the 'flashback.' A flashback is the recurrence of some of the features of the LSD state days or months after the last dose.³ A flashback occurring without apparent cause can be very frightening and cause the user to believe that he is becoming psychotic. In some individuals this concern has caused fear and depression leading to suicide. While there is some question whether LSD in itself can cause mental illness in a previously very stable individual, there is little doubt that LSD can play a role in bringing about acute and sometimes long-lasting mental illness in susceptible persons.

A number of investigators have been studying the effects of LSD on chromosomes. No conclusive or direct link has been found between LSD and chromosomal breaks, nor has it been found that such breaks will cause birth defects.⁶ Nevertheless, until further research throws more light on the question, medical authorities warn that the drug must be considered a definite risk.

Marijuana is a dried plant material from the Indian hemp plant, *Cannabis sativa*.¹⁰ For use as a drug, the leaves and flowering tops of the plant are dried and crushed, or broken into small frag-

ments which are then typically rolled into thin homemade cigarettes called 'joints', or 'reefers'.⁵ It may also be smoked in small 'roach' pipes and is occasionally incorporated into food and eaten. Hashish is the potent dark brown resin which is collected from the tops of high quality cannabis. Because of the high concentration of resin, it is often five or six times stronger than the usual marijuana, although the active drug ingredients are the same.⁶ Tetrahydrocannabinol (THC) is considered to be the basic active ingredient in marijuana and hashish.¹⁰

Marijuana has been in widespread use for several thousand years, both for its intoxicating effects and for its presumed value as a medicine. While estimates based on various surveys differ, it is generally conceded that the use of marijuana has undergone a sharp increase in the last several years, particularly among young people. The exact extent of marijuana use in the United States is not known; however health authorities believe that as many as 8 to 12 million Americans have used the drug at least once in their lives. Other estimates have ranged as high as 20 million.¹⁰ Perhaps as many as one million are 'pot-heads'. They have made marijuana a way of life, and are the equivalent of the chronic alcoholic who also uses a chemical to deal with problems of living.

When smoked, marijuana quickly enters the bloodstream and within minutes begins to affect the user's mood and thinking. The exact mechanisms of action and the alterations of cerebral metabolism are not well understood.

Because it can cause hallucinations if used in very high doses, marijuana is technically classified as a mild hallucinogen. The long-term physical effects of marijuana are not yet known. The immediate physical effects on the user while smoking include reddening of the sclera, increased heart beat, and coughing due to the irritating effect of the smoke on the lungs.¹⁰ Users also report dryness of the mouth and throat, increased hunger and sleepiness. The drug's effects on the emotions and senses vary widely, depending on such factors as the user's expectations, the circumstances of use and, of course, the strength and quality of the drug used.³ Typically, time is distorted and seems very much extended, space seems enlarged or distorted, sounds and colors can seem intensified, and thought become dream-like. The implications for the chronic marijuana user must await additional investigation.

Some tests have noted that a dose equal to one

cigarette of the weak U.S. type can make the smoker feel excited, gay, or silly. After larger amounts, the user experiences changes in perception. Colors seem brighter, his sense of hearing seems keener. After a dose equal to ten cigarettes, he experiences visual hallucinations, illusions, or delusions. His mood may become deeply depressed, or have feelings of uneasiness, unreality, or suspiciousness.¹⁰

Authorities today, are thinking in terms of drug dependance rather than addiction. Marijuana, which is not a narcotic—though for legal purposes is classified as such³—does not cause physical dependance as does heroin and other narcotics. A number of scientists think that the drug can cause psychological dependence, though, if taken regularly. All researchers agree that more knowledge of the long-termed physical, personal and social consequences of marijuana use is needed before national decisions about its legal status can be made.⁶ Researchers point out, however, that a person predisposed to abuse a drug may be likely to abuse other, stronger drugs.

NARCOTICS

The term narcotic refers to opium and to pain-relieving drugs made from opium, such as morphine, paregoric, and codeine. Heroin, Percodan, and Dilaudid are derivatives of morphine; while synthetic drugs such as Demerol and methadone are also classed as narcotics.¹¹ Narcotics are widely used in medicine as analgesics which relieve pain and induce sleep. Heroin is morphine chemically altered to make it some three to six times stronger, and since it is the narcotic used by most addicts, this discussion will focus on it.

When the abuser of a narcotic gets hooked, his body requires repeated and larger doses of the drug. Once the habit starts, larger and larger doses are required to get the same effects. This happens because the body develops a tolerance for the drug. A second sign of heroin addiction is withdrawal sickness. When the addict stops using the drug, he may sweat, shake, get chills, develop diarrhea and nausea, get running nose and eyes, and suffer sharp abdominal and leg cramps.³ There is another kind of drug dependance connected with the use of narcotics. This is known as psychological dependence. The user develops a craving for the drug for emotional reasons. He comes to depend on the drug as a way to escape facing life.

Typically, the first emotional reaction to heroin is reduction of tension, easing fears, and relief from worry. Feeling high may be followed by a period of inactivity bordering on stupor. Heroin is usually sold heavily cut or adulterated with milk, sugar, quinine, or other materials. This then is mixed into a liquid solution and injected into a vein—main-line—although it can also be injected just under the skin, or sniffed through the nose. The latter methods of use are more common among 'joy poppers' than confirmed addicts.

Heroin is a drug which depresses certain areas of the brain, and may reduce hunger, thirst, and the sex drive. Because addicts do not usually feel hungry, they can become malnourished and physically depleted. Pneumonia, tuberculosis, and venereal disease occur more frequently in addicts than in the rest of the population. The injection of contaminated material and the use of unsterile syringes and needles cause hepatitis and blood infections that may settle in the brain, heart valves, or spread throughout the body.

When the supply of narcotics is cut off, severe withdrawal symptoms may develop, including nervousness, anxiety, severe aches and pains, sweating, yawning, running nose and eyes, muscle twitch, vomiting, diarrhea and sleeplessness.⁵ Withdrawal symptoms appear in the addicted person within 12-16 hours after the drug has been last taken and become progressively worse. After 2 or 3 days they begin to subside, and within a week the junkie is free from withdrawal symptoms.¹¹ Medical authorities say that the addict is a sick person. He needs treatment for his personality problems, physical addiction and withdrawal sickness. Then, he needs considerable help to keep him going back to drug use after his withdrawal.

One promising, experimental effort to help addicts is through maintenance on methadone, a narcotic commonly used to treat withdrawal from heroin. When taken regularly, methadone eliminates the craving from heroin as well as its euphoric effects. In some neighborhoods, addicts are maintained on methadone by daily doses administered at a local community clinic.⁵ Close supervision is most important, including urine analyses to make sure that the addict is following directions about taking no drug but methadone. Modern treatment helps the addict through these withdrawal symptoms. Science now has new evidence that the body's physical addiction may last much longer than previously believed.³

Another narcotic which has begun to reappear on the U.S. drug scene is cocaine. It had been used for centuries by Andean Indians who rely on its anti-fatigue and anti-hunger effects to sustain them through a life of toil and deprivation in the rarefied atmosphere. In large doses, it produces violent stimulant, hallucinatory, and ecstatic effects. Overdoses are not uncommon and cause death from cardiac or respiratory arrest.⁸

Favorite methods of taking cocaine are by snorting and injection, or it can be mixed with heroin—speedball—to provide a smoother high.³ The effect is much shorter than that produced by speed, but is similar otherwise. The body does not develop tolerance, but marked psychic dependence results, and severe depressions occur as the drug's effects wane, impelling the abuser to continue its use. Chronic use results in nausea, digestive disorders, loss of weight, insomnia, skin abscesses, and occasional convulsions. Prolonged sniffing perforates the septum of the nose. Paranoid delusions, with auditory and visual hallucinations occur.⁸ The mental disturbances often trigger compulsive, violent anti-social acts.

CONCLUSIONS

While our emphasis here is on the public health aspects of drug abuse, there are other serious risks: the legal penalties against unlawful possession or sale of specific drugs. By becoming involved with drugs, young people gamble not only with their health but also with a possible police arrest record that may be a life-long obstacle to

a productive satisfying future.

Every incident of drug abuse is potentially dangerous, but it does not invariably lead to dependence. Even though a young person does take up drugs, the situation is not hopeless. Sometimes the main reason is to bring about a show of concern by parents. The critical test of the parents may be how they act when they find out. Parents who react with panic and recriminations only make the situation worse. This does not mean that parents accept activities which they consider harmful, but assisting and supporting young drug users in a difficult period may enable them to give up drugs. As a future member of a health care profession, it will be my responsibility to advise any young adult about potential dangers to his total health. At times, this may mean advice to him directly, or recommended referral to another member of the health care community.

Many observers see youthful drug abuse as a symptom of young people's dissatisfaction with themselves, their lives and the world as they view it. Although the so-called 'generation gap' is not unique to our time, the acknowledgement that young people have views that deserve adult attention and respect is relatively new. Studies suggest that the drug using community contains many transient users, who after a period of experimentation, discontinue drugs and re-enter the world of home, school, job, and family. Sometimes these former drug users prove to be effective educators in preventing others from trying drugs.

REFERENCES

1. "Stop Drug Abuse": National Association of Chain Drug Stores Inc. Arlington, V.A.
2. "Guidelines for Drug Abuse Prevention Education": Bureau of Narcotics and Dangerous Drugs; U. S. Justice Department, Washington, D.C.
3. "To Parents, About Drugs": Metropolitan Life Insurance Company, 1970.
4. "Resource Book for Drug Abuse Education": National Institute of Mental Health; Maryland.
5. "Drug Abuse: The Empty Life": Smith Kline and French Laboratories, Philadelphia, Pa., 1967.
6. "Stop Drug Abuse": National Association of Chain Drug Stores, Incorporated; Arlington, V.A.
7. "Sedatives, Some Questions and Answers": National Clearinghouse for Drug Abuse Information; Washington, D.C., Public Health Service Publication . . 2098.
8. "Stimulants, Some Questions and Answers": National Clearinghouse for Drug Abuse Information; Washington, D.C., Public Health Service Publication . . 2097.
9. "LSD, Some Questions and Answers": National Clearinghouse for Drug Abuse Information; Washington, D.C., Public Health Service Publication . . 1828.
10. "Marijuana, Some Questions and Answers": National Clearinghouse for Drug Abuse Information; Washington, D.C., Public Health Service Publication . . 1828.
11. "Narcotics, Some Questions and Answers": National Clearinghouse for Drug Abuse Information; Washington, D.C., Public Health Service Publication . . 1872.

Sickle Cell Anemia and its Related Visual Anomalies

by Jeffrey Dutch, Class of 1975

CHARACTERISTICS OF THE DISEASE

Sickle cell anemia is the single most neglected health problem in our country today. It effects more than one of every ten Negroes in the United States.¹ Sickle cell anemia exists in several allelic arrangements (see table). It has evolved as a result of a genetic mutation in Africa centuries ago which reduces the susceptibility to the malaria parasite. The heterozygous state, S.A., so-called sickle cell trait, effects 10% of the American Negro population, the homozygous state, S.S., effecting 0.4%, while 0.2% manifest sickle cell hemoglobin C disease, S.C., The remaining allelic states effect smaller percentages.²

The sickling process begins at birth though most infants are protected by fetal hemoglobin, F, until about four months of age. By then most of the fetal hemoglobin is replaced with the A.S, or one of the other hemoglobins. Normal hemoglobin is composed of two alpha peptide chains. Substitution of the amino acid glutamic acid in the sixth position by valine results in S hemoglobin, and by lysine results in C hemoglobin. The altered hemoglobin now causes the red blood corpuscles to become crescent-shaped or sickled, rigid, and unable to change shape. These altered corpuscles are now unable to carry oxygen and also tend to become impacted in arteries leading to vascular occlusion. Progressive damage to the deprived tissues results.³

A period of sickling is referred to as a crisis. These crises are common to the homozygous state, S.S., of sickle cell disease. The crises are characterized by sore bones and joints, abdominal pain, fever and jaundice as well as leg ulcers, spleen enlargement and hypertension. Since S hemoglobin constitutes 76-100% of the R.B.C. hemoglobin, severe anoxia may occur in an important organ or system causing multiple lesions of

the central nervous system. As a result of such complications, over 50% of the population afflicted with this disease die before the age of twenty and few live to be forty, usually crippled or disabled if they do.⁴

Sickle cell trait is usually the benign state of the disease, those showing this trait usually acting as carriers. However, under special environmental conditions such as high altitudes, infections, surgery, or under anesthesia, crisis may precipitate. An example is the death of four recruits undergoing basic training at Ft. Bliss, El Paso, Texas, which is at a relatively high altitude. The abnormal S hemoglobin count in sickle cell trait is 22-45%, so that clinical complications in sickle cell trait are generally much reduced over the homozygous state.⁵

OCULAR ABNORMALITIES

A multitude of ocular abnormalities may result from any of the allelic states of this disease, with the exception of the homozygous state, C.C., of hemoglobin C disease. Ocular lesions stem from disturbances in the retinal, disc, and conjunctival vasculature with retinopathies occurring in 33% of the population that this disease claims as victims. Dilatation and tortuosity of the retinal veins is common to all allelic states of the disease and edema may range from the optic disc to the macula. Vitreous hemorrhages are very common to the S.C. state of the disease as well as S.S. state, and although sickle cell trait fundi may show minor or no changes, complications such as exudates, retinitis proliferans, and bilateral central retinal artery occlusion have been recorded. Choriorretinal scars and obliteration of peripheral vessels have shown up in the A.C. allelic state, retinal detachments cited secondary to retinitis proliferans in the S.C. state, and black sunbursts

and lipid deposits in the S-thal state.⁶

"Since a multitude of physiological factors may affect the sickling process, it is difficult to assess the vulnerability of a given organ to sickle cell pathology. Nevertheless, it is interesting to note that the retina possesses a high rate of glycolysis, which favors a rapid depletion of oxygen from circulating red blood corpuscles. This may be an important factor in the precipitation or augmentation of sickling within the retinal capillary bed. Furthermore, the presence of an end-arteriole system in the retina predisposes to anoxia and tissue damage, once blockage occurs."⁷

RESEARCH

Although research of this dread disease has been sorely neglected, some advances have been made. Although early attempts using alkalis and antihistamines failed to halt the sickling crises, recently a team headed by Dr. Robert M. Naldbandian of Memorial Hospital, Grand Rapids, Michigan, has reported some success with a new treatment. They have found that urea, produced in the human liver, in a solution of an invert sugar destroys bonds which string the sickled cells together, and reverses the sickling process, returning the cells to normal shape, resulting in control of the painful crises. Through daily doses of the solution it is hoped that future crises can be controlled as well. Initial trials on twenty-five patients in Grand Rapids proved favorable.⁸

While these important findings are promising, experts in the field of molecular chemistry do not predict a cure for the disease in the near future. It has been reported that less than 30% of the Negro population in the United States have heard of sickle cell anemia.

Education of the population and genetic coun-

seling of the individual are necessary steps in preventing the spread of sickle cell anemia.

TABLE 1

<i>Allelic arrangement</i>	<i>Allelic State</i>	<i>Condition or disease</i>
AA	homozygous	normal hemoglobin
SS	homozygous	sickle cell anemia
AS	heterozygous	sickle cell trait
CC	homozygous	hemoglobin C disease; no sickling
AC	heterozygous	hemoglobin C trait; no sickling
SC	heterozygous	sickle cell hemoglobin C disease
S-thal	heterozygous	sickle cell thalasemia disease
SF	heterozygous	sickle cell disease with persistent fetal hemoglobin

From R. B. Welch & M. F. Goldberg, Arch. Ophth. (Chicago) 1966, 75:353-362.

- ¹ "Paradox of Neglect", T. V. 38, Worcester, MA, Jan. 26, 1972.
- ² R. B. Welch and M. F. Goldberg, "Sickle Cell Hemoglobin and It's Relation to Fundus Abnormality", Archives of Ophthalmology (Chicago) 1966, 75: 353-362.
- ³ Ibid, p. 354.
- ⁴ Archives of Ophthalmology, 85:485-490, 1970.
- ⁵ Ibid, p. 487.
- ⁶ G. Goodman, L. von Sullivan, M. G. Holland, "Ocular Manifestations of Sickle Cell Disease", Arch. Ophthal. 57:655-682, 1957.
- ⁷ Ibid., p. 676.
- ⁸ Time, 96:41 Dec. 21, 1970.

Bibliography

- W. F. McCormick, "Abnormal Hemoglobin II: The Pathology of Sickle Cell Trait", American Journal of Medical Science 241:329-335, 1961.
- R. B. Welch, M. F. Goldberg, "Sickle Cell Hemoglobin and It's Relation to Fundus Abnormality", 75: 353-362, Archives of Ophthalmology, 1966.
- J. J. Kennedy, C. B. Cope, "Intraocular Lesions Associated With Sickle Cell Disease", Archives of Ophthalmology 58: 163-167, 1957.
- B. Kabakow, S. S. van Weimobly, H. A. Lyons, "Bilateral Central Retinal Artery Occlusion", Archives of Ophthalmology 54: 670-676, 1955.
- G. Goodman, L. von Sullivan, M. G. Holland, "Ocular Manifestations of Sickle Cell Disease", Archives of Ophthalmology 58: 655-682, 1957.
- G. Pinnaas, "Macular Hemorrhage in Sickle Cell Trait with Glaucoma", Annals of Ophthalmology, Feb.-Mar. 1970, 474-476.
- S. R. Jones, R. A. Binder, E. M. Donowho, "Sudden Death in Sickle Cell Trait", New England Journal of Medicine 282: 323-325, 1970.
- F. B. Livingstone, "Anthropological Implications of Sickle Cell Gene Distribution in West Africa", American Anthropologist 60:60: 533-562, 1958.
- "The Paradox of Neglect" (Television Special) Channel 38, Worcester, Massachusetts, Jan. 26, 1972.

Federal Relations

SENATE PASSES S. 3248, INCLUDES

OPTOMETRY AMENDMENT

The Senate has passed S. 3248, the National Housing Act Amendments of 1972, which includes FHA-insured group practice facility loans for optometry. The original bill excluded optometry; however, the Senate Committee on Banking, Housing and Urban Affairs accepted an amendment in executive markup which provided the inclusion of optometry through the following language: "The term 'group practice facility' means a proprietary facility, or a facility of a private nonprofit corporation or association, for the provision of preventive, diagnostic, and treatment services to ambulatory patients in which patient care is under the professional supervision of persons licensed to practice medicine in

the State or, in the case of optometric care or treatment, is under the professional supervision of persons licensed to practice optometry in the State. . . ."

Language of the amendments, if enacted, would represent a major expansion in that proprietary facilities were not previously eligible for FHA-insured loans under Title XI in the earlier legislation. The \$5-million specific dollar limitation would be replaced with a construction mortgage limit of 90% of replacement costs (including equipment) and 90% of the sum of the cost of rehabilitation plus the appraised value of the property before rehabilitation.

WHERE ARE THEY NOW?

(This column is devoted in each issue to the effort of keeping our mailing lists up to date. Please notify Dr. Morris L. Berman, Secretary, MCO Alumni Assoc., 424 Beacon St., Boston, Mass. 02115 if you have the present address of the alumni below:)

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